MANAGEMENT OF ACUTE RENAL FAILURE COMPLICATING MASSIVE TRAUMA*

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ACUTE renal tubular degeneration as a late complication of massive trauma is increasing in frequency. An important factor in this increased incidence is the striking improvement in resuscitative measures which are now generally available and permit a significant number of severely injured patients to survive long enough to develop renal complications. The prevalence of this condition was demonstrated during World War II when 40 per cent of a group of battle casualties which required 15 or more pints of blood for resuscitation developed acute renal failure with a 90 per cent mortality rate[1]. Early diagnosis, followed by adequate surgical and medical therapy, can probably reduce this mortality figure to 50 per cent or less. This report will present the program of management practiced by our renal failure team and will offer some recent experimental data on the pathogenesis and possible prevention of this syndrome.

The early diagnosis of impending renal tubular damage is based on the recognition of diminished urine volume. Oliguria is arbitrarily defined as a 24 hour urine volume below 400 ml. or, in a more practical sense, under 15 ml. per hour. A correct diagnosis of renal tubular damage cannot, however, be made solely on the basis of oliguria since the dehydration which accompanies large crush injuries will limit urine output. This dehydration results principally from the sequestration of large unmeasurable volumes of extracellular fluid into the crushed tissues and makes any arbitrary rule as to fluid requirement impossible. Since the therapy of dehydration oliguria and of renal shutdown are diametrically opposed, it is essential that this distinction be made as early as possible. In this regard, it is helpful to have an indwelling urethral catheter inserted as soon as the patient is seen and to have hourly urine volumes

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recorded on the chart. Although there may be some risk of introducing infection into the urinary tract, with possible pyelonephritis as a sequela, we feel that the necessity for accurate, hour by hour information on the volume of urine output outweighs all other considerations in the severely injured patient. If the hourly volume falls below 15 ml., a decision must be made as to whether intravenous fluids should be restricted or forced. The answer to this problem depends on the over-all state of hydration of the patient, a factor which often can be assessed only by a therapeutic test. It is our practice under these circumstances to administer a solution of half normal saline in 2.5 per cent glucose at a rapid rate until either the urine volume rises or minimal signs of edema appear. Although any edema is undesirable, it may be practically impossible to rule out dehydration as a cause of oliguria following massive trauma without producing minimal clinical evidence of overhydration. If minimal signs of edema do develop in the absence of increased urine flow, the rate of fluid administration is sharply reduced and the therapeutic regimen for renal shutdown begun. Early, rapid rehydration will not only provide a quick diagnostic test but may possibly modify or even circumvent the clinical course of renal tubular degeneration.

The clinical picture of acute renal failure following trauma resembles, but is quantitatively different from, that associated with other types of renal failure. As a result of the crushed tissue, the NPN may rise at four times the rate and serum potassium at twice the rate for simple anuria. In addition, the increased catabolic rate which is associated with the metabolic response to injury results in an augmented rate of potassium release from uninjured tissue. We have seen the serum potassium rise 2.5 mEq./L in 24 hours in a severely injured patient.

A further complicating feature in this group of patients is the high incidence of serious infections which result from the combination of the uremia with its attendant lowering of resistance and the presence of necrotic, crushed tissue. Indeed, it is these infections, rather than uremia itself, which account for the bulk of the mortalities following renal shutdown.

The special features just discussed suggest the cardinal principles of the therapeutic program. These aim at limiting the rapid increase in blood NPN and potassium levels as well as lessening the chance for severe infection by early, radical debridement of all necrotic or crushed tissue, and also being prepared to handle the accelerated increases of these substances by means of appropriate medical therapy or, if necessary, hemodialysis.

As soon as the diagnosis of renal tubular degeneration has become established, the rate of fluid administration must be adjusted so as to balance the rate of insensible loss. At the present time, there is no adequate method for accurately determining either the amount of fluid loss, especially in a febrile patient or the rate of catabolic, endogenous water production. There are, however, two helpful guides for assessing the water balance of the uremic patient. Most important is the daily body weight which should show a steady fall of about one pound per day. A second helpful guide is the constancy of the serum sodium. If there is no unusual loss of this ion from the gastrointestinal tract, then its concentration in the serum will reflect any change in the volume of extracellular water. In the usual crushed patient, 300-400 ml. of added water per day will suffice. It must be emphasized that the route of administration, whether by mouth or by vein, is unimportant. Pulmonary edema will develop just as surely from oral as from intravenous excess. This fluid should be given as a 50 per cent glucose solution in an effort to reduce protein catabolism as much as possible.

The rapid rise in serum potassium which follows severe trauma poses a number of special problems in management that differ sharply from the usual case of non-traumatic acute renal failure. The normal catabolic rate in uninjured anuric patients will produce a rise in the serum potassium of no more than 0.5 mEq./L in a 24 hour period, but this figure may rise to 2.0 mEq./L per day or more following extensive muscle crushing. The toxic effects of this process are accentuated if a simultaneous drop in the serum sodium is allowed to develop since the physiologic effects of high serum potassium are due in part to the ratio of this ion to other positive ions. It has been demonstrated in the dog that the electrocardiographic patterns associated with serum potassium levels as high as 8-10 mEq./L can be reversed if the serum sodium is simultaneously elevated to abnormally high levels. We do not suggest deliberate elevation of the serum sodium as a therapeutic procedure, but these studies do suggest that the maintenance of the sodium levels within normal limits may be of value.

The intravenous administration of glucose and insulin has been shown to lower temporarily the serum potassium level since potassium is utilized in the transport of glucose across the cell membrane. Unfortunately, the reduction in serum potassium is of short duration and therefore the use of glucose and insulin has little place in the therapy of prolonged elevations. It is occasionally helpful as an emergency measure to protect the myocardium while the patient is being prepared for hemodialysis but should not be considered as definitive treatment. In addition, this technique has the marked disadvantage of driving the potassium into the cell where it is inaccessible for removal by dialysis.

The oral or rectal administration of exchange resins will extract potassium from the body in sufficient amounts to remove the potassium which results from normal protein catabolism. They can be considered as the definitive treatment for hyperkalemia associated with anuria in the non-traumatized patient, and will be quite adequate in the crush syndrome once the potassium load which is released from the damaged tissues has been controlled and intestinal function has resumed.

Hemodialysis is an essential therapeutic modality in the severely crushed anuric patient, both for the control of marked hyperkalemia during the first days after injury and also for the subsequent removal of nonprotein nitrogen. The indications for dialysis thus fall under two rather separate headings. Evidence of potassium and other electrolyte abnormalities as judged by serum levels, electrocardiographic changes, and skeletal muscular activity, may require dialysis within the first 72 hours of anuria. Indications for dialysis during the later period of renal shutdown are not as clear-cut and require considerable experience to evaluate. Elevation of the NPN, *per se*, is a useful guide but cannot be unreservedly accepted since following trauma, the cerebral effects of uremia will occur at lower serum NPN values than in the non-traumatized uremic individual. The most helpful clinical indication is the first evidence of disturbed psychomotor or neuromotor function expressed either as increased irritability or as somnolence. Alternate periods of depression and bursts of anger at nurses and doctors over trivial matters are ominous signs, heralding the early onset of convulsions, respiratory paralysis, and coma. One of our patients who was dialyzed on seven occasions learned to recognize the symptoms and requested that he be returned to the artificial kidney.

It is apparent that successful management requires a medical-surgical team experienced in the application of all these measures. With this combined program, the mortality for uremia following trauma has been reduced to 50 per cent in a series of 36 cases. Uncontrollable infection,

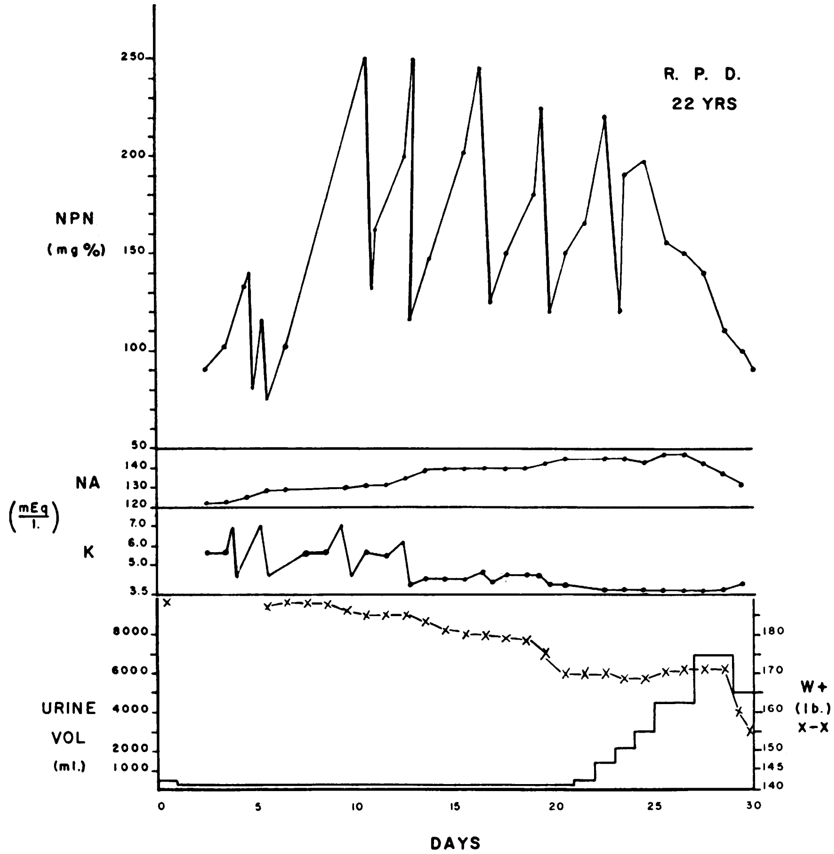

Fig. 1—Chemical data from a patient with post-traumatic acute renal failure leading to 21 days of anuria. The sharp falls in NPN coincide with the seven hemodialyses. The first three dialyses were carried out primarily for hyperkalemia. (See ref. 2. Reprinted by permission of the New York State Journal of Medicine.)

hemorrhagic diatheses, and wound healing complications were the principal causes of death.

These principles can be illustrated by a brief case report. A 22 year old truck driver was crushed beneath his overturned cab and brought to the hospital in profound shock. Because of the signs of massive intra-abdominal hemorrhage, he was taken to the operating room where emergency thoracotomy was performed because of cardiac standstill. Laparotomy was then carried out with resection of a ruptured spleen, exteriorization of the avulsed left colon, ligation of the severed inferior mesenteric vein, and ligation of the transected left common iliac vein,

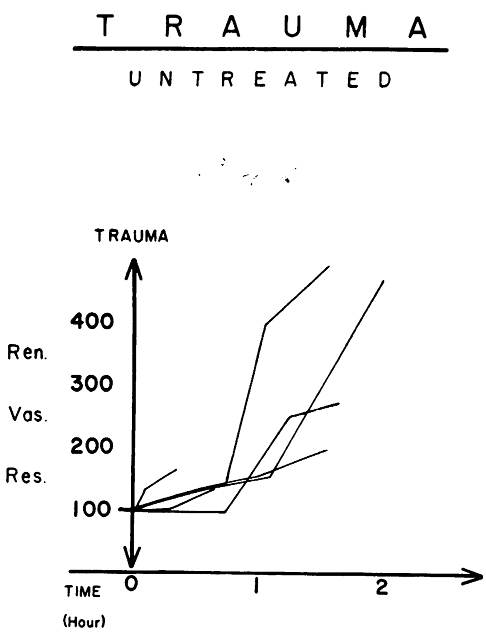

Fig. 2—Proportional changes in renal vascular resistance following mallet trauma in six animals. The control value in each case was arbitrarily taken as 100. Blood pressure in all animals remained within 10 per cent of control values. The sharp increases in renal vascular resistance indicate a proportional fall in renal blood flow.

It was noted at the time of surgery that extensive muscle crushing was present, involving the abdominal muscles, left psoas major muscle and especially the muscles of both legs, but without fracture. Sixteen hours after surgery the diagnosis of renal shutdown was established and medical therapy instituted. Figure 1 illustrates the pertinent features of the course during the succeeding 21 days of uremia. It is apparent that although the fluid intake during this period averaged only 350 ml. per day, he was overhydrated as evidenced by his final weight following diuresis. The indications for the seven separate hemodialyses are shown by the serum potassium and NPN levels. It can be seen that the first dialysis was required on the fourth post-injury day because of a serum potassium level of 7.0 mEq./L associated with marked electrocardiographic changes. His course was complicated by pneumonia, markedly retarded wound healing, and finally the development of multiple large

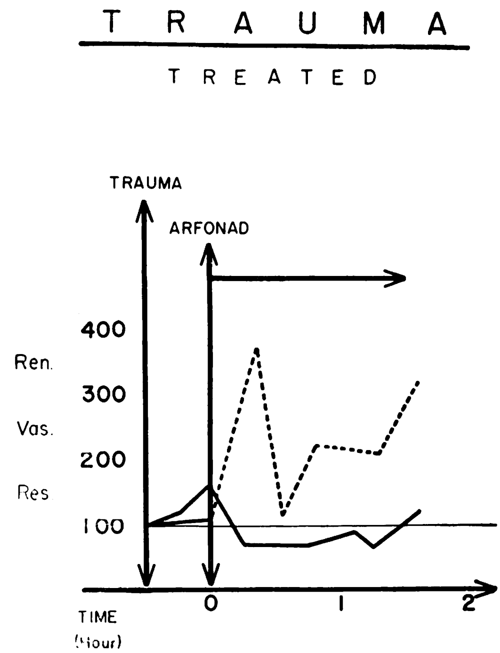

Fig. 3—Changes in renal vascular resistance resulting from the administration of Arfonad after the completion of trauma. The solid line was calculated from an animal in which the blood pressure was kept near 85 mm. Hg. The dotted line was calculated from an animal with a blood pressure which fluctuated between 30 and 60 mm. Hg. Extreme hypotension apparently produces an effective increase in renal vascular resistance (dotted line), whereas ganglionic blockade with only moderate hypotension (solid line) abolishes the effect of trauma on the renal vascular resistance.

abscesses in the crushed muscles of the thigh. It is believed that the early radical surgery, combined with frequent, properly timed hemodialysis, was an important factor in his eventual recovery. Decisions on all phases of management were made in consultation between the internist and surgeon. This case has been reported elsewhere in detail[2].

The prevention of acute renal tubular degeneration following trauma is currently under investigation and some preliminary results from animal studies are of interest. The thesis that renal tubular anoxia is an important feature in pathogenesis is generally accepted. We set out to determine whether this tubular anoxia resulted solely from the hypotension associated with shock, or whether a decrease in renal blood flow due to a selective renal vasoconstriction independent of the systemic blood pressure, might so lower renal tubular blood flow that tubular anoxia and subsequent cellular degeneration would occur. In order to test this hypothesis, graded muscle trauma was produced in the hind leg of 36 dogs. At the same time renal blood flow was measured directly by inserting a "T" cannula in the renal vein and quantitatively diverting renal venous blood into a graduate cylinder. Figure 2 shows the results of six experiments and confirms the hypothesis that renal blood flow may decrease sharply without concurrent hypotension. It is of particular interest that five of the six animals showed histological changes of acute renal tubular degeneration at the time of sacrifice two days later.

Having established that both the pathological and physiological changes of renal tubular degeneration could be produced by muscle trauma in the intact anesthetized normotensive dog, the experiments were repeated in the same way except that the kidney was completely denervated. In this group of animals the renal blood flow remained at control levels throughout the study, indicating that the renal vasoconstriction observed in the first group was mediated through the autonomic nervous system. This last observation suggested a possible technique for the prevention of renal tubular degeneration. In a third group of animals, a ganglionic blocking drug (Arfonad) was given intravenously immediately following the trauma. Figure 3 shows that although the renal blood flow decreased, this change was proportional to the fall in systemic blood pressure and therefore the effect of trauma on the renal vascular resistance had been abolished. Histologic study of the kidney, 48 hours later, revealed no evidence of tubular degeneration in seven of eight animals studied.

This experimental work is still in a preliminary stage, but it suggests the possibility that the complex chain of disturbed physiologic events which leads to renal tubular damage may be made reversible by a combination of adequate resuscitative measures and specific pharmacologic agents.

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